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Comorbidity and Taxometrics

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In the functional mental disorders, where conjointly definitive pathophysiology and etiology are unknown or nonexistent, a disease entity is specified syndromically, by the statistical relations of signs, symptoms, traits, tests, and other data. The very concept of comorbidity becomes problematic, and its application to individual patients is difficult. Coherent cut kinetics, the taxometric method composed of multiple procedures (e.g., MAMBAC, MAXCOV, MAXSLOPE, MAXEIG, L-Mode) developed by Meehl and colleagues, is urged as a partial objective solution. The importance of the INUS condition, a concept from philosophy of science, is explained. Four situations of putative comorbidity are defined with explanations of how taxometric results would distinguish them.

Key Words*: comorbidity, diagnosis, nosology, syndromes, taxometrics *[Clin Psychol Sci Prac 8:507-519, 2001]

Despite widespread uncritical use, skeptical clinicians and theoreticians hold that the concept of comorbidity in psychopathology is not in satisfactory shape, and some even doubt its meaning and possibility. More than 2,700 articles on the topic from 1995 to 1998 (a rough count from PsychLit by Scott Lilienfeld, personal communication, March 22, 1999) have not settled the matter. An excellent clarification and criticism was made by Lilienfeld, Waldman, and Israel (1994). Yet the six invited commentaries on Lilienfeld et al. surely did not resolve, or dissolve, the problem posed by their critique. I depart from the Lilienfeld et al. conclusions in holding that a nonpathology, nonetiology taxon is autonomously definable by syndrome taxometrics and, so defined, permits a purely syndromic question of comorbidity to be asked. To analyze the problem rationally and conduct definitive empirical research on it, one must clarify (a) the meaning of the term "disease entity"; (b) how, in general, one identifies a disease entity; (c) the meaning of comorbidity in general; (d) what statistics are appropriate to determine it, in general; and (e) from items c and d, how you decide comorbidity for an individual patient.[1]

The general understanding of these matters is pretty clear in the case of organic disease, often so clear that no complicated statistical method is required. In what follows, I do not assume that the medical model will or will not apply in psychopathology (nor, for those disorders where it does apply, in what ways it should be modified). I usually avoid using the phrase "medical model" because it is rarely either theoretically illuminating or clinically helpful. Most psychologists do not seem to have thought through what they mean by it or why they think it is good or bad. However, in this context it seems useful to begin with its standard theoretical meaning to get the discussion going.

I presuppose a framework of methodological and substantive assumptions that space constraints do not permit me to defend, elaborate, or exemplify properly here (readers unfamiliar with my writings are referred to Meehl, 1973, 1977, 1979, 1991, 1997; Meehl & Golden, 1982). Disease entities in organic medicine are characteristically diagnosed by a conjunction of facts: history (including family history), syndrome (symptoms and signs), and course (including response to therapy). These different sorts of facts I call indicators. They are

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taken as indicators of an inner state, the pathology, which, when we know enough about the disease, is often associated with a specific etiology. In contemporary medicine, laboratory tests, biopsies, and exploratory surgery give the physician direct access to a portion of the pathology—what is abnormal about a body tissue. A disease entity as a concept (applied to a class of patients) is defined (temporarily) by its pathology if the etiology is not known, and by a conjunction of the pathology and etiology if both are known. If two etiologies produce similar pathology and hence similar indicator patterns, two different diseases are defined by the two different etiologies (e.g., in the *International Classification of Diseases* [*ICD*]). Because of the custom that a disease entity is defined jointly by the pathology and etiology when both are known, it may happen that the manifest indicators produced by two different diseases (having the same etiology but different pathologies) may barely resemble each other syndromically. On the other hand, two diseases with different etiologies, if they have sufficiently similar pathologies, may manifest similar syndromes.

When medicine began to define disease entities by their pathology and etiology, it became evident that the usual textbook delineation of a given disease by a conjunction of history, syndrome, and course, together with pathology and etiology, required conceptual modification for a simple, obvious, and undisputable reason—namely, a patient may have the pathology and etiology of a disease without presenting signs and symptoms. Hence physicians introduced the concept of "silent," "latent," or "subclinical" disease. The clinico-pathological conference—the famous Cabot method, now routine in all teaching hospitals—educates medical students, residents, and physicians by presenting them with the clinical diagnostic problem minus the pathologist's report, the latter being presented at the end of the conference. The pathologist's report is the gold standard we psychometricians like to imagine. It is well-known that errors of omission (failing to infer a disease found at postmortem) are statistically more common than errors of commission (diagnosing a disease whose pathology is not found). A courageous report by an eminent Minneapolis diagnostician (Peppard, 1949), imposing very strict standards with regard to patients who came to autopsy, classified his various sources of error. He found that for errors of omission, 29% could not have been classified as judgment errors or inadequate reexamination on the physician's part, the reason for the omitted diagnosis being simply symptoms or signs not found. Some hyperoperational psychologists (who reject the concept of subclinical disease as unscientific) could benefit by attending clinicopathological conferences. An analogous example in psychopathology is the patient who develops florid schizophrenia at age 30, but who was not at age 28 indistinguishable in all respects, internal and external, from normal individuals. Some diseases may be latent for months or years because the chain of causation involved in the pathology is such that the slowly developing morbid changes are below the threshold of syndrome production. Specifying impairment or harm as essential to a disease taxon also is complicated by silent pathology (although this may be an uninteresting puzzle in semantics, hence fixable).

Even in organic medicine, the concept of specific etiology, when closely examined in its quantitative aspects, becomes a bit complicated. I have examined this concept elsewhere (Meehl, 1977), presenting conceptual and mathematical definitions of varying degrees of strong causal influences without regard to whether we are discussing organic or functional disorders.

When we move into the functional behavioral disorders that dominate in the field of psychopathology, this relatively clear set of metaconcepts becomes murkier. I set aside the problem of specifying what one means today by a functional behavior disorder. If forced to explain, I would say that its pathology is mostly a matter of deviations in acquired behavior (due to learning); and that if it has, in some suitably refined sense, an organic substrate (perhaps even an organic-specific pathology) that pathology is either something about the fine structure of the central nervous system (e.g., density of dendritic spines on certain kinds of cells in the limbic system) or an aberration in neurochemistry. For functional entities, the specific etiology may be a mutated gene or a statistical accumulation of adverse polygenes, or environmental factors (e.g., rejective mother, sexual trauma, substance abuse, head trauma) acting on a normal genome. Probably the most common etiology is an adverse social learning regime or environmental trauma acting upon a genetic predisposition (major locus or polygenic or both), but this causal structure is usually best classified not as specific etiology but as an INUS condition, discussed below. I take it as obvious that a genetic-specific etiology operates via a mediating link in the causal chain—that is, a microstructural or neurohumeral cerebral pathology (there being, so far as I am aware, no *tertium*

quid). There are some slightly arbitrary but, I trust, not whimsical semantic conventions to be set up here. But I do not think any of the statistical suggestions that follow hinge upon exactly how one delineates "coarse brain syndrome" ("organic" in the ordinary, old-fashioned sense of the neurologist) from genetically produced fine structure or neurochemistry aberrations.

When we contemplate the kinds of etiologies that lie behind functional disorders such as schizophrenia or manic-depression, to the extent they are genetic, it is hard to imagine any place to look except cerebral fine structure or aberrated neurochemistry. We know for schizophrenia, however, that nongenetic factors play a significant role in determining who falls ill, since the concordance for monozygotic twins is not, in recent studies, greater than 50%. Unless one believes in witchcraft or sunspots as contributors to mental illness, it follows that the cooperating factors must lie in environmental circumstances pre- and postnatal. It is convenient (Meehl, 1990) to divide the latter into the social reinforcement schedules of the developmental period (e.g., a schizophrenogenic mother, a sexually abusing stepfather) and adult, current stressors (e.g., losing a job, desertion by a spouse). Whatever may be one's theoretical conjectures and clinical practices associated with particular functional disorders, it is important to keep in mind that in functional disorders what corresponds to pathology in internal medicine is psychodynamics (broadly conceived), and that the psychodynamics are the behavioral and psychical processes and dispositions that result from a learning history imposed upon higher order genetic predispositions (Meehl, 1972). It is sometimes helpful to conceptualize this as the difference between the content of learned behavior (especially social behavior) and the parameters (mainly genetic) of the behavioral acquisition and activation functions (Meehl, 1986).

The concept of taxon has been explicated in three ways, sometimes by a combination of them. First, it is the intuitive, commonsensical way in which we use familiar expressions like "species," "syndrome," "type," "disease entity," "natural kind." A taxon is a nonarbitrary class. All taxa are classes, but not all classes are taxa. We invoke Plato's criterion that categories should "carve nature at its joints." These rough verbal synonymies are supplemented by concrete examples from the relevant empirical domains. The notion that post-Galilean science, in focusing on quantification, eschewed all taxonicity is plainly false: witness the central roles of taxa in astronomy (planet), physics (neutron), mathematics (quadratic), chemistry (iodine), geology (igneous rock), zoology (chipmunk), botany (daffodil), physiology (hormone), medicine (measles) (for more examples of taxa found in different areas of human knowledge, see Meehl, 1992).

This sort of rough-hewn delineation of the taxonicity concept is adequate for many purposes. But when categorization becomes difficult, as when we discuss *formes frustes* in mental illness, when deciding whether to be "lumpers" or "splitters," and when dealing with the present issue of comorbidity, rough delineation does not suffice. The identification of each taxon with its specific etiology, if such exists, is better. The trouble with that in psychopathology is that we do not know enough about most of the alleged entities. Even if clinicians or theoreticians believe there is a distinct taxon (such as schizotypy, or schizotaxia), they are not in a position to prove the pathology and etiology that they are speculating about. Moreover, it is an empirical fact—not something due to our carelessness, laziness or ignorance, but a fact about the world in this domain—that the syndromes are loose and the connections of the syndrome facets, or of the syndrome with the history and course, are stochastic, sometimes with only a modest degree of strength. In such circumstances, it is possible but unlikely that mathematically unassisted clinical experience can suffice to clarify matters, as it has not succeeded in doing so with respect to comorbidity. Hence, in most functional psychopathology, we have to turn to the third way of determining taxa, on the basis of the manifest indicators.

For psychopathologies that are not taxonic, it is unclear what could be meant by the term comorbidity, other than a partial overlapping of the familiar conventional lists of symptoms and signs. In the absence of taxonicity, such overlapping should lead simply to locating the patient in a dimensional hyperspace, as discussed below. I am inclined to doubt that the concept of comorbidity is theoretically clarifying or clinically useful for nontaxonic entities; and although I do not assert that, and nothing in what follows depends on it, I shall confine my statistical suggestion to taxonic situations. This suggestion includes, necessarily, the crucial matter of distinguishing taxonic disorders from nontaxonic. We do not, as rational clinicians, ask whether the patient has both disorder A and disorder B unless we have corroborated the conjecture that two such disorders exist (other than in our heads).

In organic disease, ascertaining the pathology and etiology underlying a loose syndrome reassigns some cases and provides the way to formulate a comorbidity criterion in the diagnosis of individuals. Mathematics may help but are not needed if the syndrome is fairly tight. Hundreds of diseases have been identified leading to trustworthy clinical rules for diagnosing individuals without formal statistics, and this still goes on to some extent. But for functional disorders with no pathology and etiology known (or, if alleged to be known by some, not agreed on by many others), the simple approach of the internist and neurologist is not available to us, and thus statistics of some kind are usually necessary. I do not say always. In this article I aim at dealing with the hard cases where disagreement persists. Even if we found Seymour Kety's hypothetical "purple spot" in chromatographic analysis of schizoid serum or cerebrospinal fluid, it does not follow that we would automatically become crystal clear about a proposed nosological explication in those terms. The single indicator sign positives and sibling taxon proportions under plausible models of schizophrenia will probably not be overwhelming, even if the purple spot were two-way pathognomonic with respect to a dominant schizogene (for a discouraging numerical example, see Meehl, 1999).

ENVIRONMENTAL MOLD TAXA

Using the formal numerical definition of taxonicity results in a large number of behavioral taxa that are environmental mold types (Cattell, 1946), rather than having as their specific etiology a germ, a gene, a dietary deficiency, or a specific traumatic life event such as childhood sexual abuse or a brain injury. We must resist the temptation to assume that environmental mold taxa are syndromically looser than the medical kind. I do not know any research showing that they tend to be looser syndromes, nor can I think of any strong theoretical basis for such an assumption. The empirical domain with the largest number of taxa is biology, where there are, for example, over 800,000 species in the class Insecta alone. In psychology, I suppose it is safe to say that the largest number of taxa is found in occupations, the *Dictionary of Occupational Titles* (U. S. Department of Labor, 1991), which has more than 20,000 entries. The taxon "neurosurgeon" is without doubt a clear, strong type characterized by a closely knit cluster of verbal information, sensory and motor skills, emotional stability, courage, social role, title, directory listings, self-concept, and so forth. It is widespread popular perception that computer programmers are different sorts of people from automobile salesmen. These environmental mold taxa, stemming from long and complicated stochastic sequences of life events and personal decisions, rarely have a specific etiology, even if that phrase is used loosely. My favorite example of an environmental mold taxon is Trotskyism (Meehl, 1992, 1999), a statistically very closely knit political ideology taxon defined by a pathognomic conjunction of two beliefs: "The Soviet Union is the only workers' state and must be defended against everyone, USA included," and, "Stalin is a counterrevolutionary bureaucrat who is deforming the workers' state." From this conjunctive diagnostic sign some dozen other beliefs and attitudes could be infallibly inferred (e.g., in Nazi Germany, "no enemy to the left"; "Leon Sedov was assassinated in a Paris hospital"; "Bukharin's last speech in the 1938 Moscow show trial was in Aesopian language"; "Socialism in one country is impossible"). This syndromic interknitting is far tighter is than found in most organic diseases and certainly more so than anything we find in psychopathology. But a person can become a Trotskyist for a list of completely disparate reasons (e.g., being a disillusioned Stalinist, having a lover who won't marry unless you convert, being a Norman Thomas socialist who comes to feel like a useless "parlor pink," being a Minneapolis truck driver whose successful strike of 1934 was led by the Trotskyists).

There is a weak analogy to the comorbidity question in dealing with environmental mold taxa, as if one were to ask, "Can one person be both a Troskyist and a Freudian?" The answer is yes, whereas it was extremely rare for a Stalinist to be a Freudian, as the Stalinists considered psychoanalysis to be a bourgeois pampering of the rich. To a political scientist or student of the history of ideas, such a question could be deemed worth asking and answering, although it lacks the practical importance that comorbidity may have for the healing arts. It is an interesting question why political and religious ideologies can often show a tighter interknitting of their elements than we find in organic disease or nonideological personality types.[2]

INUS CONDITIONS

A kind of causal influence that is extremely common in the life sciences (biological or social), is what philosophers call an INUS condition (Mackie, 1965, 1974; Meehl 1977, 1990). The acronym stands for *i*nsufficient

but *n*ecessary component of a causal complex, which complex is *u*nnecessary but *s*ufficient to produce the result. For example, if an output category "mental deficiency" is defined psychometrically as IQ < 70 plus inadequate life coping, one of the large number of causal complexes that can produce that outcome is phenylketonuria. The input complex has two components, dietary intake of phenylalanine and homozygous recessive phenylketonuria mutation. The mutation is insufficient to produce the result without phenylalanine in the diet, and phenylalanine in the diet is insufficient without the mutated gene resulting in the absence of the enzyme that metabolizes phenylpyruvic acid. Thus, the conjunction of these two factors is a causal complex sufficient for the defined result. But that complex is unnecessary because there are many other causal complexes that can produce mental deficiency. Each of these two factors, the gene and the dietary phenylalanine, is insufficient for mental deficiency. But each is a necessary component of the complex that is itself, while unnecessary, sufficient. Identifying an INUS condition requires a general (usual, normal, standard) background—called the "standing conditions" or "causal field"—which is normally presupposed. For example, one cannot develop Huntington's disease without adequate food and shelter to permit survival into the morbidity risk period, but we do not mention that in discussing the etiology of Huntington's.[3] As another example (from Mackie, 1974), a factory burns down due to melting of a defective fuse box that ignites a tub of waste lying under it. Because the factory could burn down without either of these factors (e.g., by arson, or being struck by lightning), each factor is unnecessary and the complex is unnecessary for a fire. But each of them is necessary if everything else is held constant, including the presence of the other. The fire requires oxygen, but since oxygen is regularly present in the atmosphere, this is a standing condition, part of the causal field that is presupposed and therefore not usually mentioned.

From these definitions, it is obvious that whether or not something is an INUS condition depends jointly on how broadly the presupposed causal field is defined and on the semantic extension of the named effect. In the phenylketonuria example, suppose the effect (outcome, result) is defined not broadly as mental deficiency, but as the more detailed outcome syndrome including pathognomonic urinary odor, fair complexion, and other aspects directly reflective of the pathology. Then the complex gene plus phenylalanine diet becomes a necessary condition, and neither of the two factors is an INUS condition under that narrower specification of the outcome category.

When, in a case of joint causality, one of the causal factors is regularly present, we tend to label the other one as the specific etiology. Strictly speaking, phenylalanine in the diet and the phenylketonuria gene have equal importance in the causal system. But since all diets in all cultures contain the critical level of phenylalanine (unless specifically manipulated because of the diagnosis), we focus on the gene as the specific etiology. We call a disease "hereditary" when, given the usual causal field, the difference between the sick and the well is genetic rather than environmental. This choice of language comes down essentially to the relative frequency with which the cooperative factors are present in the population (Meehl, 1972). One sometimes hears it said that since heritability is a statistical concept referring to a specified breeding population (more exactly, a genome-*cum*-environment population), we cannot meaningfully speak of the heritability of a disorder or trait in a single individual. Even geneticists sometimes say this, but it is clearly false. The child Isobel, found living at age 6 with her deaf-mute mother in a dark attic, tested at idiot-level IQ. After exposure to a normal environment, her mental age increased at a far faster rate than normal, and then fell to the normal growth rate at age eight, that is, in two years, suggesting her genetic level was around IQ 100. She graduated from high school at the usual age (Jensen, 1998). It is appropriate to infer that the severe mental retardation reflected by her initial IQ score was wholly environmental in origin. On the other hand, when a child born to a Jewish professional couple has idiocy-level IQ due to the homozygous Tay-Sachs recessive gene, the retardation is wholly hereditary. These clear-cut examples demonstrate that we can usefully speak of heritability of a disorder for an individual. It is unfortunate that so few psychologists are familiar with the philosopher's INUS condition concept because the majority of behavior disorders probably involve causal factors that are INUS conditions.

TAXOMETRIC PROPOSAL

For some 25 years, my colleagues and I have been developing, evaluating, and applying a set of distinct taxometric procedures for researching situations where one deals with conjectured fallible indicators of a

conjectured latent taxon. No criterion in the conventional sense of concurrent or predictive test validity is available, so that the role of, say, a psychiatric chart discharge diagnosis is merely to help get a statistical foot in the door. This is a simple point, involving both the epistemology of theories and the question of pragmatic utility. The psychoclinician is not usually in the situation of an industrial psychologist whose whole purpose in validating a test is to predict performance (e.g., which candidates will wash out of flight training). To treat psychiatric diagnosis as more than the initial stage of statistical bootstrapping or laboratory experimenting, making the diagnosis the criterion in the conventional sense, would mean that I give an MMPI or a Rorschach or compute taxometric statistics from a set of signs and symptoms for the purpose of predicting the psychiatrist's verbal behavior. This is a pointless exercise, a kind of psychometrician's parlor trick, since if I want to know what Dr. Smith is going to label a patient, the easy way to do it is to walk down the hall and ask. The nosological chart label "schizophrenia" in contemporary research is mainly a search device for locating pedigrees at risk, whose members, sick or well, can be studied with psychometric, psychophysiological, neurological, and biochemical indicators of the schizotaxic genome.

The conjectured latent structure is that we deal with two taxa, whether defined by specific etiology or by the formal numerical criteria of taxonicity; preferably they would be defined by both, but only the latter is a necessary condition. Clinicians conjecturing a latent taxon may differ among themselves as to whether they think there is a specific etiology, because the formal numerical meaning of taxonicity deliberately leaves that possibility open, not presupposed. In what follows, I also conjecture that each taxon has a base rate $P \geq .10$ (although there is evidence that my taxometric procedures may be able to detect smaller base rates). Each taxon is discriminated by quantitative indicators of sufficient range, the taxon and its complement being separated by at least one standard deviation (at least 1.5 *SD* for the MAMBAC procedure) on each indicator. Finally, we must have a sufficiently large sample for the analyses. I advocate several hundred, and in some situations, at least a thousand. One should not commit taxometrics with small samples.

Each of the procedures involves studying the statistical behavior of one or more output variables (conjectured indicators of a latent taxon) in subsets of cases specified by sliding cuts on input indicators. "Input" and "output" are used here only in the statistical sense, analogously to the independent and dependent variables in calculating a correlation coefficient. No causal imputation is intended. In fact, it will ordinarily be the case that all the indicators are causally linked as outputs to the specific etiology (if any). The generic phrase we use to characterize the method, coherent cut kinetics (CCK), means that we study what happens when a cut moves (kinetics) and that the structural and quantitative inferences made from these results should agree (be coherent). Since the several procedures subsumed under the generic taxometric method are mathematically nonredundant, such agreement functions as a corroborator, a Popperian "risky test" of the taxonic conjecture. The method, when properly used, is therefore (in a sense) self-validating. We cannot validate such a method against the clinician's judgments treated as a criterion in the conventional sense because the clinician's judgments of something like comorbidity (or even the clinician's reliability of diagnosis of individual cases in general) is precisely what is being investigated. I can think of no way to validate (I would prefer the word "appraise" or "evaluate") my taxometric method, except by extensive Monte Carlo investigation, which has now been done in numerous laboratories, or in the context of thoroughly understood organic disease, where we do have the pathologist's report. If we perform taxometrics on clinical data (history, signs, symptoms, and course), deleting the laboratory tests and the pathologist's report, we can ask whether the taxometric method gets at the truth. This indirect approach to validation using real data pseudoproblems, also called plasmodes, has at this time not been adequately explored.

Some dozen nonredundant taxometric procedures have been devised and their statistical properties investtigated to varying extents, but I confine myself here to the best known ones, MAMBAC and MAXCOV. The MAMBAC procedure (Meehl & Yonce, 1994) uses two indicators at a time, plotting a graph of the mean difference on the output variable between cases lying above and below a sliding cut on the input variable. If the generating structure is taxonic, this graph is humped; if nontaxonic, it is dish-shaped. Each indicator pair can be run in either direction, thus when 3 indicators are available the procedure yields a panel of 6 MAMBAC curves; with 4 indicators one has a panel of 12 curves.

The MAXCOV procedure (Meehl & Yonce, 1996) uses three indicators at a time, plotting the graph of the covariance of the two output variables' covariance in successive class intervals on an input variable. If the

generating latent structure is taxonic, the graph is peaked; if the underlying structure is nontaxonic, the graph is flat. Using all possible input-output combinations, we get a panel of 3 MAXCOV curves to contemplate; 4 indicators yields a 12-curve panel.

For both procedures, the location of the taxonic peak depends jointly on the taxon base rate P and the separation of the latent input means. For a given separation (crude latent taxonic validity), and assuming that all indicators are keyed such that the taxon scores higher on each indicator than does the complement class, the peak location depends on P and shifts to the right as P declines.[4]

It is preferable that candidate indicators for a conjectured taxon should be antecedently chosen on the basis of clinical experience, nontaxonic quantitative research, or theory. That is, we prefer confirmatory to exploratory taxometric analysis. However, that is not always possible, and some investigators prefer to avoid it.[5] When a selection of candidate indicators is not theory driven, for whatever reason, the TAXSCAN procedure is available (Meehl, 1999). TAXSCAN consists simply of sequentially scanning an ordinary correlation matrix so as not to bother with pairs and triads that are negligibly intercorrelated, followed by using CCK procedures on correlated variables. The suggestions for taxometric solution of the comorbidity problem that follow apply to either the confirmatory or exploratory situation. I consider five cases which we want our taxometric analysis to distinguish as to their structure and the latent quantitative parameters and, having reached those conclusions, to provide as accurate a method of diagnosing individuals as the validities permit. I shall assume that appropriate consistency tests are performed and will not discuss them.[6] In the ideal case, what I am about to say concerning the shapes and their changes will hold for all or almost all of the single curves generated by the taxometric procedures.

As always, being a scientific realist, I formulate the situation ontologically rather than epistemologically. What I say about the taxa and the indicators is what is known to Omniscient Jones and not (initially) known, perhaps not even conjectured, by the investigator. In formulating the mathematics of the objective taxonic structure, we of course talk about how things really are, rather than about how the clinician thinks they are.

Case I. True Comorbidity

There are independent taxa A and B, and a complement, C. For instance, A might be schizophrenics, B manic depressives, and C a mix of other psychiatric patients, or normals, or both. Quantitative indicators (x, y, z) are indicators of taxon A, they distinguish it from both B and C. Quantitative indicators (u, v, w) are indicators of B, distinguishing it from both A and C. Approaching our data atheoretically, we would use TAXSCAN (Meehl, 1999) on the entire sample and thereby identify a cluster of pairwise correlations among (x, y, z) and another cluster of pairwise correlations of (u, v, w). We detect six taxonic MAMBAC curves among the (x, y, z) pairs and, as this finding leads us to expect, three taxonic MAXCOV curves for this triad. The same holds for the triad (u, v, w). We conclude that there are two taxa, A and B, and a complement C. Rearranging our table of intercorrelations based on what we have learned, we note clusters of negative correlations in the cells defined by the cross-taxon pairs; that is, r_{xu}, r_{xv}...r_{zw}, the nine parataxonic correlations, are negative and further corroborate the existence of two taxa (Meehl, 1999).

Next we define a sequence of subpopulations (still using the entire sample) by a sliding cut on the simple unweighted sum ($x + y + z$) of standard scores of the variables that discriminate taxon A, and we do MAMBAC and MAXCOV again on (u, v, w) for the sequence of subpopulations. As this cut moves to the right (from a "pure" complement of taxon A, through a mixture, to a "pure" taxon A subgroup) nothing happens to the taxonic shape of the (u, v, w) triad curves because taxa A and B are independent. The base rate of taxon B does not change as we move the cut. The expected comorbid rate on the independence assumption is the product $P_A \times P_B$ of the two base rates. We may also arrange a sequence of subpopulations defined by the Bayes' Rule diagnostic probabilities of individuals, which will give results similar to the sliding ($x + y + z$) cut. If we make estimates of the valid and false positive rates achieved by the hitmax cuts for each indicator, the incidence of various sign-patterns, on the assumption of comorbid independence, can be computed. We can also define subpopulations by ($u + v + w$) and check for changes in taxon A within them.

Case II. One Real Taxon, the Other Illusory

One source of disagreements about comorbidity is that clinical experience or nontaxometric research may lead to a belief in a taxon which does not exist but is suggested by a cluster of pairwise correlations that are correct but generated by a nontaxonic factor. This case is easily identified because real taxon A is identified, as

above, by the coherent MAMBAC and MAXCOV graphs of (x, y, z), whereas the correlated pairs (u, v, w) in the TAXSCAN matrix will be found to be nontaxonic when analyzed by MAMBAC and MAXCOV. In such a case, I am inclined to think that the question of comorbidity is meaningless, so I do not consider it further.

Case III. A Taxon with Subtaxa

The latent situation is the existence of a taxon A which has subtaxa A_1 and A_2 and a complement class, C. For example, A could be schizophrenia, A_1 paranoid schizophrenia, and A_2 nonparanoid schizophrenia, an old suggested dichotomy that some clinicians still accept, and which cannot be determined syndromically (or genetically, lacking direct access to DNA etiology) without taxometric analysis. In this situation, schizophrenic indicators (x, y, z) discriminate A from C, and they also discriminate A_1 and A_2 from C. Indicators (u, v, w), the paranoid indicators, discriminate subtaxon A_1 from subtaxon A_2 and they also discriminate A_1 from C.

Here again, if we define a sequence of subpopulations by a sliding cut on the sum $(x + y + z)$, or by Bayes' rule of diagnostic probabilities as based on that triad, as the subpopulations defined approach a new base rate $P_A \rightarrow 1.0$, the MAMBAC and MAXCOV graphs for the triad (u, v, w) remain clearly taxonic. Because the base rate of the paranoid subtaxon A_1 is rising by the reduction in C (moving toward a subpopulation consisting wholly of schizophrenics), the base rate P_{A_1} rises, approaching whatever is the base rate of paranoids among schizophrenics.

If we move the $(x + y + z)$ cut downward or define successive subgroups by progressively lower Bayes' rule of schizophrenic diagnosis, our proportion of $A \rightarrow 0$. The (u, v, w) peak of the taxonic graphs shifts to the right because these subpopulations, having almost no schizophrenics, have almost no paranoid schizophrenics; and finally the peak becomes simply a high point at the upper end, which, for sufficiently low values in the schizophrenia rate, disappears, the (u, v, w) curves becoming nontaxonic in shape. High confidence cases of subtaxon A_1 are almost all members of A, so the proportions of (x, y, z) configurations are predictable by taking $P_A = 1$.

If we consider the hexad (x, y, z, u, v, w), its 30 MAMBAC and 60 MAXCOV curves will behave taxonically because all 6 of these indicators discriminate the paranoid schizophrenics from C. Our overall estimate of the base rates within the whole group will of course show

$$P_{A_1} < P_A$$

$$P_{A_2} < P_A$$

$$P_{A_1} + P_{A_2} = P_A .$$

In medicine it often happens that a clearly identified disease may arise as a complication of another disease (e.g., renal failure in diabetes). Although the alternative etiologies of the secondary disease entity are distinguished and of clinical importance (for prognosis, therapy), from the taxometric standpoint one could subsume this under either case I or case III. We could opt to call paranoid delusions with hallucinations a complication found in some cases of schizophrenia, instead of the conventional subtype; taxometric analysis would not distinguish the two verbal conventions. This illustrates the general point that taxometric findings, like other statistics, are not self-interpreting as to causal relations.

Case IV. No Real Taxa but an Appearance of Such

The obvious way this case can arise is from a latent nontaxonic structure in which two or more nontaxonic factors, quantitative and unimodal, have markedly different factor loadings on subsets of candidate indicators. Dimensional factor A strongly loads indicators (x, y, z) but loads indicators (u, v, w) weakly. Factor B loads indicators (u, v, w) strongly but (x, y, z) weakly. Clinical experience will therefore present two groups of patients, one characterized by an (x, y, z) syndrome and the other by a (u, v, w) syndrome. But what the clinician notices are merely cases occupying a subregion of the descriptor space, which need not be clumped in order to be selectively attended to. The clinician has seen something real, but it is not a taxon.

There are other more complicated causal situations that will lead to a clinical conjecture (for less skeptical clinicians, a conclusion) that there are taxa when there are not. For instance, because of some theoretical bias, a clinician may expect to see an (x, y, z) clumping in the descriptor space and hence will erroneously believe that clinical experience confirms this (e.g., Chapman, 1967; Chapman & Chapman, 1969, 1975). Psychometric artifacts due to unusual distribution of test scores or rating scales may lead to clumping in the descriptor space which, although empirically real, reflects the unusual statistical properties of the metric rather than a latent taxon. On the other hand, the rarity of certain combined deviations, especially cases reversing sign of the overall correlations, may capture a clinician's attention and induce a false taxonic inference. It may happen that

the social or clinical importance of a certain combination of quantitative traits sensitizes the clinician to notice them.

My generation of Minnesota clinicians were educated by our mentor, Starke Hathaway, and his psychiatric colleagues to a strong belief in the existence of psychopathic personality, asocial, amoral type (old nomenclature), and we had the impression "You've seen one, you've seen 'em all." Some recent unpublished work casts doubt upon the existence of the psychopathic taxon; hence I no longer have that degree of confidence, although I'm still betting on it. It could be that the strong impression, shared by many clinicians, that there is such a taxonic entity arises from the fact that these patients are in some ways so unlike most other psychiatric patients (e.g., not anxious, less depressed, extrapunitive in reaction to frustration), plus their being of special significance socially because of their externalizing orientation and danger to others. Finally, they are notoriously refractive to conventional psychotherapy, form no transference bond, enter into no therapeutic alliance, and tend to fail appointments and not pay the bill. The nontaxonic latent possibility here is that these patients are extreme deviates as to low anxiety parameter with low social fear, resulting in weak *n Affiliation*, but show a spurious sociability and a spurious interpersonal warmth due to their lack of normal social fear, resulting in weak superego development, judgment defect, and an inability to learn by experience. I would be inclined to add two (perhaps orthogonal) dimensions, high energy level and Sheldon's "mesomorphic toughness." A taxonic finding in this fuzzy domain should be carefully scrutinized as to the indicators, lest a socially defined syndrome of delinquency be conflated with the purely psychological personality type one has in mind.

Whatever the origin of the erroneous conjecture, the CCK method will reveal its erroneousness. As I said previously, I do not know how to formulate the idea of comorbidity if there are no taxa to be comorbid. If extended to case IV, what comorbidity would seem to mean is that the patient deviates positively on both of the latent generating factors, presenting a composite of six high values of the indicator set (x, y, z, u, v, w). With a weaker construal, adopted because of the usual looseness of syndromes in psychopathology, we might consider persons presenting, say, with ($x^+ y^+ z^+ u^+ v^+ w^-$) or even ($x^+ y^+ z^- u^+ v^- w^+$) as comorbid. Any such mixed syndromic criteria will be arbitrary because, unlike the taxonic case, there is no pattern of pluses and minuses for each of the two alleged syndromes that yields a diagnostic Bayes' rule probability > .50 as a decision basis. How comorbid somebody is, diagnosed on the basis of such a pair of clustered indicators, cannot be other than a matter of degree (sign counts), and its objective reference would be the patient's inferred location in the underlying factor space.

Case V. A Nonoptimal Case That Is Likely to Arise in Psychopathology for Certain Common Signs, Symptoms, Traits, or Test Scores

We might have a situation in which one or more indicators cut across several taxa, discriminating them from the general complement class but by different amounts. For example, most psychiatric patients suffer some degree of depression on interview and as shown by psychometrics (e.g., the D scale of the MMPI). Even considering groups of patients where interview or psychometric depression is conventionally considered low, this lesser degree is usually by comparison with our average expectation for the mentally ill. The hysteroid valley on MMPI, the familiar conversion hysteric profile coded 13′, deviates from the statistically more common D spike. But the *belle indifference* of the hysterical patient does not usually produce a D score at or below the general population norm; it is just that it falls below the two somatizing keys adjoining it and below the typical D score of psychiatric patients. Most somatizing patients are depressed to some degree. The same is true of the nondepressed psychopath who often has a reactive depression, although mild, from losing freedom and being in a boring mental hospital with "crazy people," offending the psychopath's pride. Patients whose formal diagnosis is major depression have the highest average scores on the D scale, but many other patients also score high on that scale. Some schizophrenics, for example, have very high D scores, as did patients diagnosed "anxiety state" in the old nosology.

Ideally, when investigating comorbidity, one seeks to find or construct indicators that one conjectures to be nosologically specific, but that is not always feasible. Suppose (x, y, z) indicate taxon D (major depression), where x = depressed mood score. But x also discriminates taxon S (schizophrenia), although less elevated in that group, which is indicated more strongly, and specifically, by measures (u, v). All five measures differenttiate the set (D + S) from the complement class C (normals, or abnormal

other than our D and S groups), but the separation $(\overline{x_D} - \overline{x_C}) \gg (\overline{x_S} - \overline{x_C})$. A TAXSCAN search will identify D and S, the indicator sets (x, y, z) and (x, u, v), each set behaving taxonically; and the x-indicator overlap will not distort estimates of P_D, P_S or the latent separations of each taxon from its complement class, composed of (S + C) for D and (D + C) for S, respectively. When shared indicator x is used as input for the taxometric procedures, the MAMBAC and MAXCOV peaks for the three outputs y, z, and yz should agree within tolerance, ditto outputs u, v, and uv; but the former peaks should not agree with the latter, except by an unlikely combination of countervailing systematic errors. In all CCK work we rely on Einstein's dictum, *Raffiniert ist der Herrgott, aber boshaft ist er nicht*. Nature is not trying to make a Dutch book against us. The larger the difference between x's validity for taxa D and S, the greater the displacements of the taxonic peaks. We have a one-way inference. Marked location disagreement occurs with two taxa being x-discriminated, but unequally, from C. But if they do match, we are less confident, and may elect to conclude nothing without further study. We may delete x from the candidate set and use MAXSLOPE (Grove & Meehl, 1993) as a two-variable (y and z) MAXCOV surrogate. Combining this with MAMBAC on (u, v) we should get the same base rates P_D, P_S, C as we got when x was included. Since base rate estimates are not corrupted by the syndromic overlap in x, the latent structure is further corroborated by the base rate relation $P_D + P_S + C = 1$, within tolerance, or challenged by its falsity. The unfavorable circumstance in which multiple taxa are all discriminated with available indicators has not, to my knowledge, been investigated.

IMPLICATIONS

My taxometric work has led some psychologists to conclude that I believe there are numerous taxa, or that most psychopathology is taxonic. That is not my view. On the contrary, I think most of the received categories in functional psychopathology do not have real existence as taxa. It is odd to conclude that somebody who invents a statistical method to distinguish between taxonic and nontaxonic factual situations must therefore believe in the preponderance of the former. Furthermore, I do not hold that if a *DSM* rubric does not denote an existent taxon in my sense, it is somehow illicit and should be dropped from the nosology. That is a deep and complex question having semiotic, mathematical, causal, and pragmatic components. It is usually treated superficially, and to consider it properly is beyond the scope of this article. Suppose research discloses that there are very few taxa in functional psychopathology, as I would confidently predict, so that practically all of the rubrics of *DSM* are like case IV. How should the psychopathology diagnostic system be revised? The obvious answer is that factor analysis should be used to identify the important underlying factors, and patients should then be diagnosed by specifying their location in the multiple factor space, rather than by subsuming them into categories that do not denote anything except regions in the space that clinicians somehow choose to attend to. There are two difficulties with this proposal. First, not quite everybody is contented with multiple factor analysis as a procedure, even when it is of the confirmatory kind that has credibility because it takes a Popperian risk. To discuss that issue properly is beyond the scope of this article and my competence. The second objection to a thoroughly dimensional approach involves the pragmatics of communication and clinical application. That is also somewhat beyond my scope here, but it is perhaps appropriate to say a few words about it.

As to clinical decision-making (e.g., which antidepressant to try first), there is at present no clear proof, even in the case of genuine taxonicity, that therapeutic indications will be more accurate when decisions are mediated via subsumption of the patient under a particular rubric rather than by a statistically optimal combination of the indicator variables. We are accustomed to taking for granted that we need a mediating category label, by analogy with organic disease; but that is not known to be true, and there are mathematical considerations and Monte Carlo evidence that suggest the contrary (Grove, 1991). This is a problem of great importance which has been unaccountably neglected by both physicians and psychologists. Empirical as well as further analytic and Monte Carlo investigation is much needed. If a taxonic sorting is not strongly incremental for the pragmatics of clinical decision over direct statistical combination of the observational variables used to arrive at it when a taxon exists, *a fortiori* categorizing patients under a rubric that does not represent anything but a region in the descriptor or latent factor space is unlikely to be useful and might be harmful.

It is hard to justify routine use of diagnostic categories, either for research or clinical purposes, on grounds of handy communication if what is communicated tends to mislead. But one could develop

a communicative language that would be terse and easy to remember without a terminology wrongly implying objective taxonicity. The widely used MMPI code provides a nice example. Clinicians who routinely use the MMPI learn to speak of patients in terms of Hathaway's code (1947), as when we say, talking of a psychopathic-like profile, "This patient is a 49′" and other sophisticated MMPI users know immediately what we are talking about. I know from our conversations that Hathaway, in inventing the code, had no intention of claiming that the patients showing a certain code must belong to a distinct disease entity. He was open as to which multiphasic scales or profile patterns were suggestive of a real categorical entity and which were probably not, and, for the most part, he had little interest in that question. Hathaway did, on one occasion, opine that the elevation of an MMPI scale score purported to indicate the probability of the patient's belonging to the nosological group, rather than the amount or intensity of a trait, such as dominance or social introversion (W. G. Dahlstrom, personal communication, June 21, 1999). Present-day routine usage of the code tells us nothing about the taxonic conjectures of the users, and I do not even have an educated guess as to how many MMPI users think that codes 49′ (psychopathic deviate) or 27′ (depression, anxiety neurosis, phobia) or 31′ (hysteroid personality) are related to objectively real taxa. Of course, the convenience of such a numerical summarizing notation for multivariate profiles depends on how many variables you have to look at. I should be surprised if a factor analysis of psychopathological signs, symptoms, and test scores, based on patients in the generic complement class (that is, who do not belong to any of the few taxometrically identified categories) would reveal more than 10 factors underlying a heterogeneous set of indicators. In that case, we would be able to use the 10 digits as in the Hathaway code, and research plus clinical experience would gradually accumulate knowledge about causal theory and therapeutic indications associated with patients being located in different regions of the 10-variable factor space.

Suppose taxometric analysis reveals the existence of comorbidity for two taxa, one or both of which is characterized solely by the formal numerical taxometric structure with no reference to specific etiology or to a small number of alternative INUS conditions. Why is the question of comorbidity worth asking? Here is the only place my analysis diverges significantly from that of Lilienfeld et al., but only by virtue of the clarification taxometrics may provide. If I understand them rightly, absent something etiological (at least suggested, if not criterial) they doubt the meaningfulness or utility of asking the comorbidity question. While in the current nontaxometric way of treating it I agree with their criticism, I suggest that an adequate taxometric analysis should enable one first to show there is a taxon implicitly definable nonetiologically and without reference to the psychodynamics (which plays here the role that pathology does in organic disease); and, having so shown, setting up cutting scores and estimating sensitivities and specificities (valid and false positive rates achieved by a given cut on quantitative indicators), one can answer with respectable probability the question whether a certain patient before us is comorbid. Why would one bother asking and answering this question in the purely formal numerical situation?

One would ask the question for several reasons, pragmatic and theoretical. Without having a specific etiology (or a list of INUS conditions) in the backs of our minds—even as what philosopher Herbert Feigl used to call a "promissory note" or "music of the future"—a purely statistical taxon may be associated with practical problems of prognosis, treatment choice, dangers, and complications. When an obsessive-compulsive neurosis has resulted in extreme impairment of function in major life sectors, it may precipitate a major depression in one genetically predisposed to that illness. Major depresssion, whether unipolar or bipolar and whether spontaneous or event precipitated, implies a lifetime suicide risk of about 20%, some say even 25% if we always knew the facts. Hence arises the pragmatic question whether it is safe for such a comorbid patient to be seen on an outpatient basis, as we would with an obsessive-neurotic who was not severely depressed. As another example, a patient currently labeled "schizo-affective disorder" may be simply an atypical schizophrenia, suggesting different trial medications from a taxometrically identified patient who is genuinely comorbid. Suppose the base rates of genetic schizotaxia and cyclothymia were each approximately $P = .10$, as I conjecture—perhaps larger for the latter taxon. Then a true schizo-affective comorbidity has a non-negligible probability of .01, yielding a yet less-negligible incidence in the clinical population. Note that this question can be answered formal-numerically without regard to a particular clinician's theoretical views about the genetics of schizophrenia.

On the theoretical side, fundamental research on such important matters as the soft neurology and psychophysiology of a syndromically identified taxon will obviously benefit from such identification. Mere syndromic overlap, without taxometric identification and resulting taxometric case classification, will involve a criterion of attenuated construct validity and thereby obscure what may be theoretically important psychophysiological correlations. In the discovery process, the ability of even a seasoned, skilled, highly perceptive clinician to notice correlations is hampered if the clinical experience involves a grouping of patients into categories that ought to be split. While ideally we would like our taxa to be tied to etiologies, either specific or INUS, a taxometrically identified group based on the statistics of history, syndrome, psychometrics, and course seems to be a justifiable kind of categorization.

A cautionary (pessimistic?) note is in order. Under adverse circumstances, involving nearly total overlap of indicator lists and unfavorable statistical values (e.g., low base rates, small separations), taxometrics will probably fail to do the job, as will other latent class search procedures. Such obscured multitaxonic situations will be difficult to distinguish from merely syndromic comorbidities arising from a nontaxonic multifactorial structure. Even a single latent nontaxonic factor having variable loadings or different thresholds over several signs and symptoms can generate a high incidence of syndromic comorbidities (e.g., 51% for panic/anxiety disorder) as pointed out by Brown, Antony, and Barlow (1995). Only more direct independent access to etiological factors (e.g., neurochem.-ical analysis, documentation of objective life history event) is capable of distinguishing taxonicity from mere regions of clinical interest or artifactual densification in the multivariate descriptor space. The statistics of causally dependent variables cannot do everything for us.

NOTES

1. Defining a disease entity is not the same as how the phrases "mental disease" or "mental disorder" are defined, a meta-meta question of little or no scientific interest. Some rough definition is perhaps needed for legal purposes, third-party payments, etc., but I am not even sure about that. The question is not relevant here, since concepts of harm, disablement, distress, or reproductive fitness are not essential elements of taxonicity.

2. For example, one of my doctoral students constructed a purely face-valid inventory of religious attitude and belief by Q-technique factor analysis of a very small number of clergy and laity from four Christian denominations. I warned him that Professor Donald G. Paterson, one of the final oral examiners, would be unhappy about the lack of any external validity criterion. The student then administered his face-valid internally consistent instrument to students at Luther Theological Seminary (this was a half-century ago, before the Lutherans began to go down the drain with the other liberal Protestants) and to a student religious interest group known for its anti-institutional and anti-clerical religious views. An unweighted sum of the standard scores on his two dimensions revealed zero overlap between the two groups.

3. Speaking of Huntington's as having a dichotomous specific etiology involving an error at the "Huntington locus" is not refuted by recent research identifying a varying number of CAG repeat sequences linked to differentiated age of onset. Different levels of causal analysis, each valid at its level and not inconsistent with levels above and below, necessitate adoption of suitable semantic conventions. A dichotomous qualitative etiology may be analyzable as a disjunction, as in the Huntington example. In ordinary language, "Was the victim killed by a revolver shot" has a yes-or-no answer, but the weapon may be a Smith and Wesson 30, a Colt 45, or whatever. On the quantitative side, exposure to a germ as specific etiology involves a taxon (bacterial species), but one may press the point by asking, "How many single tuberculosis organisms suffices to induce disease?" Other cases than disjunction and number may arise, where semantic stipulations, while conventional, will have to be made in the light of factual knowledge.

4. Trull, Widiger, and Guthrie (1990) used MAXCOV to study the borderline personality and found no central peak but a high point on the extreme right; they concluded that the results were consistent neither with a taxonic or nontaxonic situation, whereas the correct conclusion is that the base rate is small.

5. Some cluster analysts avoid it on principle, insisting that every observable property on which the entities of a population show appreciable differences (e.g., wing area of honeybees) must be included in the set of candidate indicators. Discussion of that (debatable) methodological principle is beyond the scope of this paper.

6. The first obvious consistency test is simply the coherence of the results (e.g., base rates, separations) delivered by different subsets of indicators and by the two different procedures discussed here. If the investigator has doubts, there are additional procedures, such as minimizing $SS_{below} + SS_{above}$ (Meehl & Yonce, 1996, p. 1135; I am adopting the acronym SQUABAC for this procedure). Which among the dozen consistency tests available should be considered mandatory for

any purported CCK application is under current investigation. For brief exposition of several, see Meehl and Golden (1982) and Waller and Meehl (1998).

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